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Right Atrial Thrombus Formation After Transvenous Catheter Ablation of the Atrioventricular Node

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The formation of a right atrial mass was detected in a patient by two-dimensional echocardiography 3 weeks after successful transvenous electrical ablation of the atrioventricular node had been performed. The mass was attached to the atrial septum at the site where the electrode catheter used for the ablation had supposedly been located and it exhibited no mobility. It was interpreted as a right atrial thrombus induced by the ablation procedure. Although no pulmonary embolic events have been observed during a 7 month follow-up period, right atrial thromboembolism must be considered a potentially dangerous complication of transvenous catheter ablation to control cardiac arrhythmias.

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Transvenous catheter ablation of the atrioventricular (AV) node is an effective treatment for selected patients with various types of drug-resistant supraventricular arrhythmias. Complications have been observed in only a small number of patients (1–3). We report on the formation of a right atrial thrombus at the site where the AV node had been successfully ablated by this technique.

Case Report

Patient data. A 25 year old woman was referred to our hospital because of recurrent drug-resistant tachycardia with a wide QRS complex and a heart rate of 240 beats/min, which had been leading to cardiovascular collapse during the last five episodes. The patient had been treated unsuccessfully with seven conventional and five investigational antiarrhythmic drugs, including amiodarone. She had no history of peripheral venous thrombosis and had never taken drugs predisposing to a hypercoagulable state. Physical examination revealed no abnormalities. Routine laboratory tests including blood coagulation analysis, two-dimensional echocardiogram (Fig. 1) and chest X-ray film were normal. The 12 lead electrocardiogram showed a regular sinus rhythm at a heart rate of 76 beats/min with narrow QRS complexes and a normal PQ interval.

Electrophysiologic evaluation in a drug-free state, using previously described techniques (4), disclosed that the patient had AV nodal reciprocating tachycardia with functional right bundle branch block. During episodes of tachycardia the patient complained of dizziness and presyncope. Because of the previous failure of antiarrhythmic drug treatment to control the tachycardia, no further drug testing was performed during the electrophysiologic study.

Ablation procedure. After informed written consent had been obtained from the patient, she underwent transvenous catheter ablation of the AV node according to the technique described by Gallagher et al. (2) in a second procedure. After positioning of electrode catheters in the right ventricle, high right atrium and low right atrium at the region of the AV node, a bolus of 100 units/kg body weight of heparin was given intravenously, followed by an infusion of 1,000 units/h for 12 hours. Using a standard 6F tripolar (USCI) pacing electrode, two direct current shocks of 200 and 300 J were delivered to the AV node 30 minutes apart. This resulted in complete AV nodal block with a regular escape rhythm at a rate of 38 beats/min and right bundle branch block-shaped QRS complexes. Two hours after the procedure no type of tachycardia was inducible.

On the following day a DDD pacemaker was implanted uneventfully. The patient then received low dose heparin (100 units/kg twice daily) subcutaneously for the next 7 days until she was fully mobilized. Blood coagulation tests showed no prolongation of thrombin time with this dosage. On the eighth day after transvenous catheter ablation she was discharged.

Follow-up. At the first control visit 2 weeks after the procedure the patient reported no recurrences of tachycardia.

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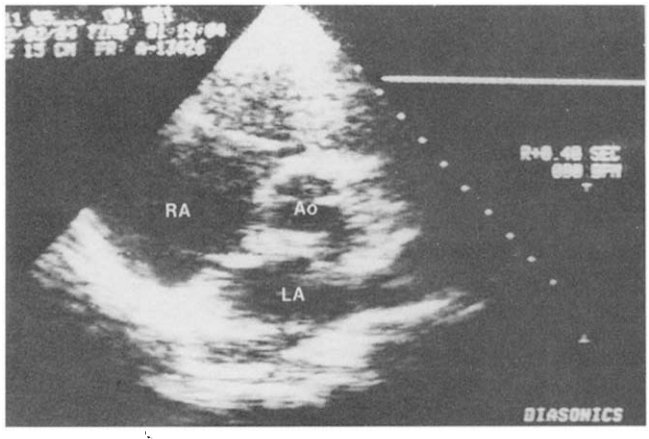
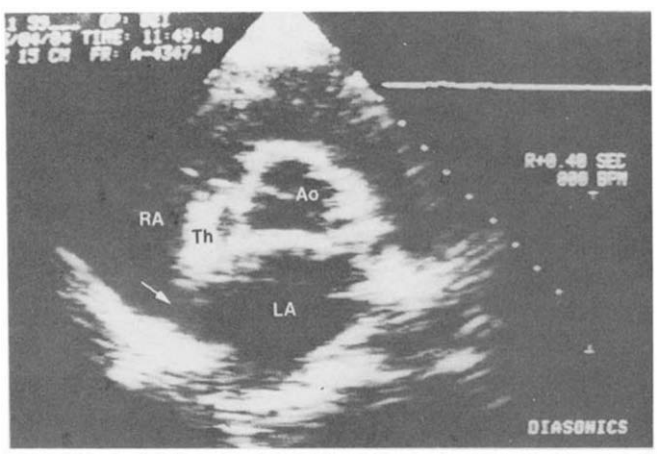


Figure 1. Two-dimensional echocardiogram in the parasternal short-axis view at the level of the aortic valve, taken before transvenous catheter ablation of the atrioventricular node. Atrial cavities are echo-free. Ao = aorta; LA = left atrium; RA = right atrium.

Physical examination showed no deviation from the first presentation. The 12 lead electrocardiogram showed only pacemaker-induced ventricular depolarizations, synchronized to atrial events.

Two-dimensional echocardiography. On the two-dimensional echocardiogram a strongly echogenic, oval-shaped mass about 25 mm long was detected in the low right anterior region of the atrial septum, suggesting the formation of a thrombus within the right atrium (Fig. 2). The mass appeared to be firmly attached to the atrial septum, from which it could be clearly differentiated in various echocardiographic views because of its higher echogenicity. It exhibited no mobility during the cardiac cycle, thereby causing no impedance to right ventricular inflow. This structure had not been observed on the two-dimensional echocardiogram before transvenous catheter ablation (Fig. 1).

Figure 2. Two-dimensional echocardiogram of the same patient as in Figure 1, taken 3 weeks after transvenous catheter ablation of the atrioventricular node. The orientation of the image is the same as in Figure 1. A highly echogenic right atrial mass (Th), attached by a broad base to the atrial septum, is clearly visualized. Note disappearance of posteromedial portion of atrial septal echo (**arrow**) because of acoustic shadowing due to thrombus. Abbreviations as in Figure 1.



By injection of indium-111–labeled platelets, an attempt was made to assess the thrombogenic activity of the thrombus surface using sequential indium-111 scintigraphy (5). There was, however, no evidence of new platelet apposition at that time. The patient was discharged on a daily oral dose of 330 mg of acetylsalicyclic acid in combination with 225 mg of dipyridamole as prophylaxis against thromboembolic complications.

During further control visits every 4 weeks the echocardiographic appearance of the right atrial mass remained unchanged and there were neither clinical nor scintigraphic signs of pulmonary embolism. Electrocardiographic controls with the DDD pacemaker in the "off" mode still revealed complete AV conduction block and a slow ventricular escape rhythm. After 7 months the patient's follow-up course has been uneventful without recurrences of tachycardia.

Discussion

In the treatment of certain drug-resistant supraventricular tachyarrhythmias, transvenous catheter ablation of the AV node is considered to be an effective therapeutic measure. However, serious complications have been observed either immediately after the procedure or during early follow-up (3). The complication of thrombus formation after transvenous catheter ablation has not yet been reported. In our patient a right atrial mass was detected by two-dimensional echocardiography 3 weeks after transvenous catheter ablation of the AV node. This mass had not been present on the same echocardiographic views before transvenous catheter ablation. It originated low anteriorly at the right atrial septum, that is, precisely where the electrode catheter used for ablation had been located during the procedure.

Differential diagnosis of right atrial masses. The mass was assumed to represent a thrombus as a consequence of direct current application to atrial tissue rather than a malignant tumor, atrial myxoma or tricuspid valve vegetation (6). A malignant tumor or atrial myxoma represents a chronic condition and should therefore have been detected before transvenous catheter ablation. A tricuspid valve vegetation is highly unlikely because of the lack of mobility of the mass in concert with valve leaflet motion. Thrombus formation as a consequence of electrical current application has been reported in animal experiments (7).

Tissue changes after electrical ablation. Histologic studies in dogs undergoing transvenous ablation of AV conduction have shown mild fibrous reaction at the base of the septal tricuspid leaflet in those dogs that had chronic complete heart block after the shocks (8).

In a patient who died 5.5 months after transvenous AV

node ablation by a single shock of 275 J (9), microscopic examination of the endocardium overlying the AV node was normal, and no inflammatory cells were present (9). In another patient, who died suddenly 5.5 weeks after transvenous catheter ablation of the AV node by two shocks of 500 J each (1), there was no evidence at necropsy of intracardiac clots and no gross evidence of myocardial damage in the region where the shocks had been delivered.

Macroscopic or microscopic findings with regard to endocardial changes provoking thrombus formation as a consequence of transvenous catheter ablation have not been described. Because of the lack of other thrombogenic factors in our case, the application of two shocks of standard energy levels to the region of the AV node had probably resulted in endocardial damage extensive enough to lead to local thrombus formation.

Sequelae of right atrial masses. Thrombus formation within the right atrium may lead to pulmonary embolism (6,10). How often emboli originate in the right atrium of patients with pulmonary embolism is not definitely known, but it is considered rare (6,11). In our patient there was no evidence that the thrombus gave rise to pulmonary complications and neither surgical intervention nor thrombolytic therapy was required. However, the outcome of patients with a right atrial thrombus may be fatal, especially if the thrombus is mobile, and physicians performing transvenous catheter ablation should be well aware of this risk.

Clinical implications. We suggest that patients undergoing transvenous catheter ablation of the AV node should undergo anticoagulation with antiplatelet drugs for 4 weeks after low dose heparinization. After that period the endocardial surface is expected to have healed. We recommend the combined antiplatelet treatment with dipyridamole and acetylsalicyclic acid rather than treatment with warfarin (Coumadin), because this combination has been shown to prevent thrombus formation in animal experiments (12) and has less potential for bleeding complications (13). Two-dimensional echocardiograms should be performed repetitively after transvenous catheter ablation to detect the potentially hazardous formation of right atrial thrombi, necessitating further diagnostic and therapeutic interventions.

Our observation indicates that, because of the relatively short duration of use and the lack of long-term follow-up data, unexpected complications of transvenous catheter ablation may still occur. Therefore, the technique should be applied with caution and only after other methods have failed.

Conclusions. To our knowledge, this is the first case of thrombus formation after transvenous catheter ablation of the AV node. Because there were no mitigating factors in our patient that might cause a hypercoagulable state, we believe that thrombus formation as a consequence of transvenous catheter ablation may not be an isolated incidence but may be a complication of transvenous catheter ablation that has hitherto not been recognized because it has not been looked for systematically.

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